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EXFOLIATIVE CYTOLOGY OF THE LARYNX —A PRELIMINARY REPORT.*

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and

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We have been investigating the possible practical value of exfoliative cytology of the larynx as an aid in early diagnosis of carcinoma.

It is a well known fact that laryngeal carcinomas sometimes are delayed in diagnosis. There may be a considerable period between the time the patient first sees a physician until the diagnosis is final. The doctor may use the approach of watchful waiting rather than alarm the patient by immediate hospital admission for formal laryngoscopy and biopsy. We hope that exfoliative cytology can shorten the delay period by giving the office practitioner a relatively easy and atraumatic method for screening suspected lesions of the larynx.

The technique used in this study is to cocainize the larynx as one would for a direct laryngoscopy and then with mirror laryngoscopy to obtain a direct smear, using the Wagner's camel's-hair brush and wiping it firmly over the suspected lesion (see Fig. 1). This brush is obtainable as a standard

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item from surgical supply houses. In taking smears from growths of the vocal cords the best results have been obtained by placing the brush between the cords, having the patient phonate, and moving the brush from anterior to posterior with the cords approximated. In extrinsic lesions it is necessary to rub the brush firmly over the lesion.

As soon as the specimen is obtained the material is painted on a labeled glass slide, and the slide is immediately dropped into a fixative solution composed of equal parts of 95 per cent

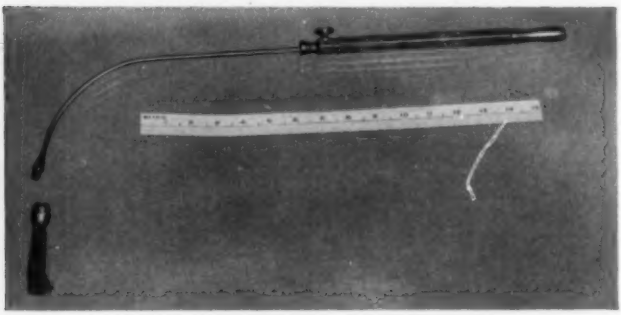

Fig. 1.

alcohol and ether. The slide must remain in this solution at least two hours, although it may remain as long as one week. After fixation the slide is ready for staining and final processing. Processing is the same as for Papanicolaou smears obtained from any other organ, using the OG-6, EA-50 stain.

In our study the slides were screened by a technician who knew the clinical diagnosis but not the biopsy diagnosis. All positive, suspicious, and questionably negative cases were examined by the cytologist, who also had no knowledge of the biopsy diagnosis.

In the first 18 months of this study 99 cases were examined. In 95 of these the cytological interpretation was checked by biopsy diagnosis. The other four cases were all

patients who had laryngoscopies performed for reasons other than biopsy, usually paralyzed vocal cords.

TABLE I. LARYNGEAL SMEAR STUDY—BIOPSY DIAGNOSES.

Laryngeal Nodule	30
Squamous Cell Carcinoma	23
Papilloma	11
Chronic Laryngitis	10
Keratosis	8
Leukoplakia	6
Granuloma	2
Cyst	2
Negative	3
	95

Table I shows the results of biopsies on the group of 95 patients. Of these, 72 had benign lesions and 23 had squamous-cell carcinomas.

TABLE II. LARYNGEAL SMEAR STUDY—CLINICAL DIAGNOSES.

Laryngeal Nodule	27
?? Carcinoma	21
Carcinoma	17
Laryngitis	13
Polyps	8
Leukoplakia	6
Papilloma	6
Cyst	1
	99

The next table shows the clinical diagnosis in this same group of patients. Here 38 were classed as either apparent or suspected carcinoma. Of the 23 proved carcinomas, 22 were included in one of these two groups. The suspected group was usually labeled "rule out malignancy." The large number

in this class indicates to some extent the difficulty in making a clinical diagnosis of early carcinoma of the larynx.

Interpretation of laryngeal smears from the 23 proved carcinomas gave 15 positive, four suspicious, and four negative, an apparent accuracy of 65 per cent. In practice it would obviously be wise to class the suspicious as at least tentative positives until biopsy can confirm the results. On this basis, the useful accuracy would be over 80 per cent.

The remaining four cases, false negatives, are not unexpected. The inherent histological characteristics of the larynx make it highly improbable that 100 per cent accuracy of diagnosis will ever be achieved—even biopsy sometimes fails. In addition, we found in the early days of our study that the examiner occasionally did not obtain the specimen from the suspected lesion. For example, one patient had an early lesion on the laryngeal surface of the epiglottis difficult to visualize. A smear taken by a first-year resident was negative; one taken at the same examination by a third-year resident was positive.

Probably the most important reason for false negatives is that it is not uncommon for carcinoma of the larynx to invade before a typical carcinoma *in situ* stage is found on the exfoliating surface. Carcinoma *in situ* is one in which the malignant cells involve the surface but have not invaded beyond the basement membrane; furthermore, some tumors are very well differentiated, and the exfoliating cells are so slightly altered that they are not distinguishable from the normal or very slightly abnormal cells frequently seen in benign conditions.

It is encouraging to find that there were no false positives in the entire group. There were, however, six cases reported as suspicious from the 72 negative biopsies. We think that these false suspicious reports may be occasioned by abnormalities of the surface epithelium, such as atrophy, hyperplasia. acanthosis, hyperkeratosis, or dyskeratosis. In any event all cases either positive or suspicious should be confirmed by biopsy. The cytological test is intended to be a screening procedure for biopsy rather than an ultimate diagnostic method.

The following three case histories have been summarized to help show the possible future value of this procedure:

The first patient, a 29-year-old white female, was first seen at our hospital 11 years ago, in 1944, when she was only 18 years of age. At that time she had lost her voice, and a lesion of the larynx was biopsied and interpreted as a papilloma. On review of this slide, we now classify this lesion as a carcinoma *in situ*. Her symptoms recurred in 1950 and a biopsy was again interpreted as a papilloma, and again on subsequent review of the biopsy we have also reclassified this lesion as carcinoma *in situ*. Her admission this year was due to two months of hoarseness. Laryngeal brush smears were positive for tumor cells, but this time the biopsy showed definite microscopic evidence of shallow invasion, in addition to the surface involvement. Total laryngectomy was performed.

This case illustrates the value of detection of a carcinoma which should have an extremely favorable prognosis.

The other interesting aspect of this case is the 11-year interval between the initial biopsy which showed no invasion and the recent biopsy which did show evidence of shallow invasion. Clerf has reported one case similar to ours, of an 11-year interval, and Stewart reported one of a seven-year interval.

The second patient, a 57-year-old white male, was followed for six months by an otolaryngologist in another city. In this case complaint was hoarseness, and the lesion of the larynx was interpreted as an inflammatory lesion by the original examiner. He made many sets of smears and cultures for acid-fast bacilli. The patient was referred to an otolaryngologist at St. Luke's Hospital, who made a clinical diagnosis of carcinoma. Laryngeal brush smears were positive for tumor cells and the biopsy showed a squamous-cell carcinoma.

Here is an example of a six-month delay due to an error in clinical interpretation. Had the original physician had a simple office procedure available, he might have been alerted sooner.

The third patient, a 56-year-old white male, entered the hospital with the complaint of hoarseness of one year's duration. Five months previously, a smaller tumor had been removed from one of his vocal cords at another hospital. This was interpreted as negative for tumor. His hoarseness continued. The clinical diagnosis on this admission was probable laryngeal nodule, but the possibility of carcinoma could not be completely excluded. The tumor was described as small and on the under surface of the vocal cord. The smears were strongly positive. The biopsy was diagnosed as squamous-cell carcinoma.

This is an example of a case in which the cytology was so strongly positive we would have asked for the biopsy despite the clinical appearance of the lesion or the history.

From these preliminary results it appears that laryngeal cytology may have particular advantages in diagnosis. Because of speed and simplicity it could be a quick office test for lesions which are clinically doubtful in nature. It could, incidentally, also do what cervical cytology has done in encouraging the physician to visualize the organ under study. Most important, because of its atraumatic nature it could be used as a serial test to follow suspicious conditions or biopsy-proved cases which have been treated by irradiation alone. In the latter, a recurrence might be detectable earlier cytologically than clinically. Another application might be in those patients whose biopsy findings were inconclusive. At present these patients require close following with frequent biopsies, an uncomfortable and expensive program. A program of frequent office visits with brush specimens taken on each visit would be less expensive for the patient and would enable the physician to add to the clinical evaluation a simple aid in follow-up care. Needless to say, if the clinical appearance of the larynx were noticeably different from previous examinations a biopsy would be in order.

This first series has included only a selected group of hospital patients. We cannot yet predict that the results will be similar with a wider range of subjects. The work is being continued to include office patients, and those who on the basis of clinical examination are not to be biopsied at the same time. Some of these patients will have such premalignant conditions as leukoplakia or atypical keratosis, and may develop a carcinoma *in situ*.

Since carcinoma *in situ* can be expected to exfoliate diagnostic cells at least in some instances, and since less than 2 per cent of laryngeal carcinomas are now diagnosed in this non-invasive stage, any additional tool to screen symptomatic patients before the development of a late lesion would be worthwhile. If, in even a small percentage of these, unsuspected positives can be detected and confirmed, the procedure will prove its value.

EFFECTIVENESS OF CONDITIONED ELECTRODERMAL RESPONSES (EDR) IN MEASURING PURE-TONE THRESHOLDS IN CASES OF NON-ORGANIC HEARING LOSS.

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The problem of non-organic hearing loss grew to major proportions for the military services during and following World War II, and it has continued to be significant in recent years, especially for the Veterans Administration. Now, with increasing concern in industry about the effects of noise on hearing and subsequent compensation claims for hearing losses, the problem of non-organic hearing loss will undoubtedly become magnified.

Relatively successful tests have been developed for the *detection* of malingering and psychogenic deafness[12,17], but these tests do not usually *measure the threshold of hearing.* For years, otologists and audiologists have looked for an "objective" test for the measurement of pure-tone thresholds, that is, a test in which responses to tone can be made independent of the will or control of the person tested, and in which the responses can be scored with a minimum of subjective interpretation. A test that seemed to hold promise of providing a measure of threshold in a relatively objective manner involved the use of electrodermal responses (EDR)* to tone as an indication of hearing.

The use of changes in the electrical properties of the skin as an indication of hearing in cases of non-organic hearing loss was reported as far back as 1915. Wiersma[44] observed changes in the electrical resistance of the skin associated with auditory stimuli to help him distinguish organic deafness

* More commonly designated as "psycholgalvanic skin response" or "galvanic skin response" and variously abbreviated PGSR, GSR, PGR, PG and GR.

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from simulated or from functional deafness. Since that date others have reported[1,11,26,29,33] similar use of electrodermal responses to tone.

Interest in this so-called "objective" test of hearing was revived by Bordley, Hardy and Richter[6] in their report of a modification of the EDR technique. They proposed that for clinical use tones be followed by an annoying shock, so that conditioned responses to tones could be established. This conditioning procedure, then, could make measurement of threshold by the EDR method more feasible, because it would not permit the nervous system of the person tested to adapt to the auditory stimuli even at low sensation levels.

Since the report of Bordley, Hardy and Richter[6], there have been numerous reports[3,6,7,13,14,20,25,31,37] describing the successful use, or the probability of successful use of conditioned electrodermal responses not only for detecting non-organic hearing losses but also for obtaining accurate audiograms in those same cases. There have also been scattered reports of the failure of the EDR test with some cases of malingering and psychogenic deafness[2,17,18,41].

The purposes of this paper are: (1) to describe the clinical experience at Central Institute for the Deaf with the EDR test as a means of measuring pure-tone thresholds in patients suspected of having non-organic hearing losses; (2) to discuss the problems involved in the EDR test with these kinds of patients.

We are not concerned here with the success of the threat of shock in the EDR test in coercing or encouraging better cooperation in conventional testing procedures. We are considering only the success of the tester in eliciting electrodermal responses to tones so that reliable auditory thresholds can be determined from those responses.

OBSERVATIONS.

Twenty patients suspected of having non-organic hearing losses have been examined by the EDR test. Some of the tests were eminently successful while in other tests the pa-

tients gave few or no reliable responses in spite of intensive conditioning. If we take into account the responses during conventional audiometry (patient asked to raise his finger when tone is heard, lower it when tone is no longer heard), as well as the responses during the EDR test, we can describe four general categories of responses during our pure-tone tests.

1. Poor conventional responses—good EDR.
2. Good conventional responses—good EDR.
3. Poor conventional responses—poor EDR.
4. Good conventional responses—poor EDR.

We considered the conventional responses to be poor when they did not occur consistently with a given level of the tone which was apparently supra-threshold, when the pure-tone thresholds were grossly inconsistent with the patient's scores on speech tests or with his obvious ability to hear conversational speech, or when the behavior of the patient indicated that he was deliberately withholding responses.

The 20 cases were about evenly divided among these four categories. The reason for the approximate rather than definite division of cases among the particular categories is the continuous rather than discrete nature of the categories themselves. For instance, those patients whose electrodermal responses were consistent but whose conventional responses were neither very consistent nor very inconsistent could be placed justifiably in either category 1 or 2.

The following examples will illustrate the range of success with EDR as outlined above.

1. POOR CONVENTIONAL RESPONSES—GOOD EDR.

A. M. was a young Air Force nurse with a history of acute aerotitis and an ensuing permanent hearing loss. Malingering was suspected, however, because of inconsistencies in audiometric scores and because of the patient's urgent desire to be separated from military service.

Chart I is a record of the patient's audiogram and speech test results.

CHART I.

In this, and succeeding charts, pure-tone and speech thresholds (spondee word lists) determined by standard procedures are given in decibels of hearing loss with respect to normal thresholds. Discrimination scores (PB max.) are given in per cent of words correctly identified from phonetically balanced word lists presented at intensities well above the patient's threshold (or estimated threshold) for speech. Pure-tone thresholds determined by the EDR test are given when they are available.

	125	250	500	1000	2000	4000	8000	Speech Thresh.	PB Max.
Right AC	70	60	65	70	75	75	90		88%
BC			40	40	40				
EDR (AC)			10	10					
Left AC	(responses only at maximum output of audiometer)								84%
BC			60	60	60				
EDR (AC)				65					

Electrodermal responses to tone alone were so consistent that no conditioning shocks were used. The patient was in tears after the EDR test had progressed for about 10 minutes. She probably realized that we were not deceived because we presented only tones which were close to normal threshold. The 10 db losses in the right ear at 1000 and 2000 cps represent the patient's maximum hearing losses at those frequencies. We might have obtained consistent responses with weaker tones had we attempted conditioning. The 65 db loss in the left ear at 1000 cps probably represents threshold of hearing in the right ear from sound conducted through the skull. We made no attempt to mask the right ear while testing the left ear.

The other four subjects in this category did not yield electrodermal responses as easily as did A. M., but conditioning of responses was relatively easy in those cases and we considered our judgments of thresholds to be reliable.

2. GOOD CONVENTIONAL RESPONSES—GOOD EDR.

M. H. was a 16-year-old girl who gave consistent and reliable responses during conventional audiometric tests. She was referred for an EDR test because of suspicion of a non-organic hearing loss for the following reasons: *(a)* the examining otologist was able to carry on a direct conversation with her with relative ease in a normal voice in spite of her poor performance during the test of speech discrimination; *(b)* her audiogram, especially for the left ear, was rather unusual: a rising audiogram with maximum sensitivity at 3000 cps is not common in cases of apparent perceptive-type hearing losses; and *(c)* the onset of the hearing loss was rather sudden, apparently only six months prior to visiting the otologist. The cause of the hearing loss could not be ascertained.

The following chart is a summary of the audiometric findings on M. H.

CHART II.

	250	500	1000	2000	3000	4000	6000	8000	Speech Thresh.	PB Max.
Right AC	35	45	45	30	5	15	65	75	67	6%
BC		45	50	30	10	25				
EDR (AC)			50		15	> 40	55?			
Left AC	40	50	50	45	35	20	55	45	71	6%
BC		35	X	X	35	30				
EDR (AC)						45?				

In that the EDR thresholds indicated no more hearing than the conventional audiogram, the conventional audiogram was considered valid, and the hearing loss was regarded as organic.

EDR testing was begun at 1000 cps in the right ear. The responses were consistent and the estimated threshold corresponded closely with the threshold determined by standard procedures. As the EDR test progressed the responses became less frequent in spite of nearly constant reinforcement. Thus, some EDR thresholds given in the chart are qualified as being uncertain. At any rate, there were no electrodermal responses to tones at less intensity than the threshold values determined by conventional procedures. Of the other four patients in this category, two gave responses as consistent as those of M. H., one gave fewer reliable responses, and one gave more frequent reliable responses.

3. POOR CONVENTIONAL RESPONSES—POOR EDR.

L. S. was a 48-year-old man who claimed compensation for serious loss of hearing in his left ear as a result of a blow to the head while he was at work.

Two EDR tests were given two weeks apart, and conventional audiometric tests were done on both days. L. S. cooperated no better during the second attempt at a pure-tone audiogram than he did during the first. His behavior during the conventional pure-tone test was characteristically that of a malingerer. By contrast, he seemed to cooperate completely during the speech tests. The following chart shows the discrepancy between the pure-tone and speech tests. No bone conduction tests were attempted. There were not enough electrodermal responses to tone to permit an estimate of threshold.

CHART III.

	250	500	1000	2000	4000	8000	Speech Thresh.	PB* Max.
Right AC	5	5	10	20	40	30	3	66%
Left AC	35	25	45	50	80	50	10	80%

* Same intensity level for both ears, approximately 45 db above normal speech thresholds.

L. S. did not fail to give clear electrodermal responses to shock. He even gave an occasional clear electrodermal response to live voice close to normal threshold. His responses to pure tones, however, were very sparse; they were less frequent during the second session than they were during the first; nevertheless, during both sessions he admitted, without prompting, that he was hearing many tones. It was almost as though he hoped that his admission would deter us from giving a shock. At no time, however, that he told us he heard a tone did he show what could even be considered as a probable electrodermal response.

We attempted to confirm that L. S. was malingering by the Lombard test, but a white noise at approximately 130 db above 0.0002 dyne/cm^2 produced no detectable effect on his speaking voice. We followed the suggestion of Hanley and Tiffany[22] in trying to disrupt reading and spontaneous speech of L. S. by feeding back his own voice to him at a high intensity and with a delay of about 0.2 second. This procedure had no effect on his speech.

Of the five patients in this group, L. S. gave the poorest electrodermal responses, but none of the others gave enough consistent responses to establish a reliable threshold in either ear at any frequency.

4. GOOD CONVENTIONAL RESPONSES—POOR EDR.

H. H. was a 16-year-old boy who gave a history of rather sudden onset of hearing loss about six months prior to these examinations. His responses during the conventional audiometric tests were consistent, and

CHART IV.

	250	500	1000	2000	3000	4000	6000	8000	Speech Thresh.	PB Max.
Right AC	50	45	55	55	60	45	50	50	58	80%
BC		25	35	45	35	35				
EDR (AC)			50?							
Left AC	40	35	40	30	20	20	35	30	28	90%
BC		15	30	40	20	40				
EDR (AC)				30-40?						

speech thresholds and speech discrimination were commensurate with the pure tone audiogram. The lack of positive otologic findings, however, along with the flighty, distrustful nature of H. H. and a story of unhappy home life all led to a suspicion of a psychogenic overlay on a possible organic hearing loss. The test results are summarized in Chart IV.

H. H. was difficult to condition, in spite of almost constant shock reinforcement. Shock was definitely annoying and elicited large electrodermal responses. A bright floodlight was used later in the test, in addition to shock, as an unconditioned stimulus, but this did not help to condition electrodermal responses to tone. The EDR thresholds given in Chart IV are uncertain; more dependence was placed on overt reactions in anticipation of the shock than on the occasional electrodermal responses.

Two of the other patients in this category gave similarly poor electrodermal responses, while two gave a few more dependable responses.

DISCUSSION.

We have reported a larger proportion of unsatisfactory results with the EDR test than is generally described in the literature. We are not able at this time to explain the discrepancy; nevertheless, we shall try to account for some of the difficulties which may be encountered in measuring pure-tone thresholds in cases of non-organic hearing loss.

A study on normally hearing children[19] indicates that there may be many completely normal people who are difficult to condition. It is probable, therefore, that ease of conditioning may not be related principally to the nature of the auditory disorder of the person tested, as had been suggested earlier[18], or to the particular conditioning procedure.

It can be inferred from the writings of James[28] and Fuller[15] that relative ease of conditioning in general may be a function of some psychophysiological characteristic of the individual and not of conditioning procedures or of some pathological state. These investigators described differences in conditioning behavior between different breeds of the same species of animals. Lacey, Siegel and Siegel[30], however, looked for some relevant morphological factor in humans, but did not find any relation between body form (height-weight ratio) and either basic skin conduction or electrodermal responses.

The single factor or constellation of factors which characterize humans who are easy to condition or difficult to condition has not been established. Welch[42], Welch and Kubis[43], and

Bitterman and Holtzman[4] did present evidence that anxiety was positively related to success of conditioning. A later study by Bitterman, Holtzman and Barry[5] failed to confirm their index of anxiety as a predictor of the degree of conditioning. Taylor and Spence[40] noted that anxiety neurotics were no easier to condition than other neurotics. Hardy, Wolff and Goodell[23], in their studies on pain, found no relation between apparent emotional tension and magnitude of electrodermal responses during a period of adaptation or habituation. We have not observed that persons who were overtly anxious in the test situation were conditioned any more easily than those who appeared calm.

Taylor and Spence[40] did find that the eyeblink response was conditioned more easily in psychotics than in neurotics, but Greenwald[21] recorded virtually the same EDR from mental patients as he did from normals under the same conditions. Paintal[35] found no impairment in psychotics of the physiological mechanism for producing electrodermal changes, but he did note that psychotics responded far less than normals to the threat of a shock.

Gantt[16], Reese[38], and Reese, Doss and Gantt[39] noted that electrodermal responses as well as other autonomic and skeletal responses were markedly more difficult to condition in psychotics with "diffuse cortical impairment" than they were in normals. Peters and Murphree[36] reported that chronic schizophrenics gave smaller electrodermal responses and were more difficult to condition than normals. They also found that a group of schizophrenic patients who had spent three months learning to solve problems "with insulin stimulated hunger as a motive" reacted more strongly and conditioned more easily than the untreated patients. If, then, we consider some cases of deafness to be symptomatic of a psychosis, we may possibly expect the EDR test to be less successful in those instances than in cases of organic deafness.

Sedatives may also limit the effectiveness of the EDR test. Depressing effects on electrodermal responsiveness by "suavitil"[27], "gardenal"[32], and barbiturates in general[24] have been reported.

Hardy, Wolff and Goodell[22] pointed out that ingestion of a small quantity of alcohol can raise the EDR threshold to an ordinarily painful stimulus by as much as 780 per cent. We can speculate that with so large an increase in threshold to painful stimuli it might be difficult to elicit responses to sounds near auditory threshold. We have had experience with two normal adults from whom we could get no responses to tone after they had a small amount of liquor. A study of the effects of alcohol should be undertaken to learn whether a well-coached malingerer could "beat the game" by drinking just enough liquor before the test so that his drinking would not be suspected from the smell of his breath or from his behavior. Similar studies might well be undertaken of the effects of barbiturates and other sedatives.

A promising clue to predicting the ease of eliciting responses and possibly to the understanding of the mechanisms involved comes from the electroencephalogram. The work of Darrow, Pathman and Kronenberg[10], and of Mundy-Castle and McKiever[34] indicates that the existence of a strong 9-11 per second rhythm (or lack of low-voltage fast activity) may be inversely related to the responsiveness of the autonomic nervous system. Our own preliminary studies[9] on normal adults in which we recorded EEG and EDR simultaneously appears to confirm these observations with respect to the ease of eliciting electrodermal responses. Electrodermal responses were difficult to elicit from subjects with a strong 9-11 per second rhythm. Our studies seem to show, in addition, that simultaneous EEG and EDR recording may be desirable, because quite often changes will occur in the EEG as a result of the tone when no EDR can be detected.

The EDR test as now administered may provide information about pure-tone thresholds in cases of non-organic hearing loss that may not be obtainable from conventional tests on the same patients. Failure of the EDR test, however, is too common to accept lack of responses as meaning organic hearing loss. If the person who administers the EDR test is aware of the limitations and shortcomings of the test, he can better evaluate the results of the test and may derive valuable information from it.

SUMMARY.

Twenty persons suspected of having non-organic hearing loss were tested by conditioning electrodermal responses (EDR) to tone, and also by conventional audiometric procedures in an attempt to establish a reliable pure-tone audiogram. The responses during those tests fell into the following four categories with approximately equal distribution of cases in each category:

1. Poor conventional responses—good EDR.
2. Good conventional responses—good EDR.
3. Poor conventional responses—poor EDR.
4. Good conventional responses—poor EDR.

The distribution of the easy to condition and difficult to condition patients does not differ clearly from the distribution of normally hearing, cooperative adults and children.

As it is currently used in cases of non-organic hearing loss the EDR test can, with many patients, provide information which other audiometric procedures fail to produce. The EDR test fails sufficiently often, however, so that lack of electrodermal responses to tone cannot always be equated with an organic hearing loss.

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HEADACHE—A PROBLEM OF MODERN OTOLARYNGOLOGY.*†

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INTRODUCTION.

Because some of the pathways that we in otolaryngology have been travelling seem to have reached blind endings we have partially and inadvertently accepted the idea that otolaryngology as a specialty is dying. Although many old ideas and many old practices are dead, a time has arrived not for burial but for re-evaluation — the beginning of a Renaissance.

The otologist if he is to survive, henceforth must be an otophysiologist, a neurotologist and a better otologic surgeon than he ever was to meet today's demands. The laryngologist must be a wide-field neck surgeon, a competent enough neurologist to know that the larynx may be painful without local pathology, and an astute student of the causes of speech and voice defects which have their origins far higher than the hyoid bone or the nasal sinuses. The rhinologist has ceased to operate for sinus infections that never existed. He now understands where the effective realm of nasal surgery lies and what its proper relation is to the nose as a physiologic organ of special sense and respiratory function. He is re-evaluating the relation of gross anatomy and pathology to micro-anatomy, physiology and neurology.

It is with such an altered viewpoint that we must approach the problem of headache.

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THE BASIC MECHANISMS.

When a patient complains of headache he may be speaking of any pain in or about the head or neck. Behind this symptom complex there are, grossly, two mechanisms. The purely sensory mechanism involves only the pain sensory tracts. These convey pain sensation from their source in the periphery directly to the sensorium, or by reference they may project sensations of pain to terminals at a related but remote point. Thus an inflamed lower tooth may convey its message via the mandibular division of the Vth nerve directly to the sensorium to be identified as an aching tooth; or it may by reference to the auriculotemporal branch of the mandibular nerve be projected to its terminals to be identified not as a toothache but as an earache. The pain of an inflamed maxillary sinus supplied by the maxillary division of the Vth nerve may be referred to the area ordinarily associated with the frontal sinus which is of course supplied by the first division of the Vth nerve.

The second or vascular mechanism consists of the pain sensory tracts originating in the blood vessels of the head and neck. These also may convey pain sensations directly to the sensorium, or by reference they may project them via the peripheral sensory system to a remote point; thus edema of the wall of the carotid artery may cause severe pain at the site of the edema. The enlarged and painful vessel may be seen and palpated, and it may be mistaken for an enlarged painful lymph gland; or the pain from this vessel may be referred to the shoulder and arm of the same side.

In explaining the vascular mechanism of headache, Wolff[1] postulates by experiment that vascular headaches are caused by distention of the extracranial vessels, distortion of the venous sinuses, or distortion, displacement, or dilation of the intracranial arteries.

By recording and comparing the immediate effects of intravenous injection of histamine on the cranial and extracranial arteries he concludes that histamine headache immediately following the use of histamine is the result of stretching of the intracranial vessels. The explanation, however, does not seem to apply to the delayed clinical histaminic cephalalgia described by Horton.

Hilger[2] has clarified the explanation of the mechanism of vascular pain by showing that spasm of the vasa vasorum of an artery produces intrathecal edema of that vessel. This edema stretches its coats and so produces pain as surely as does the actual vasodilation postulated by Wolff. Thus the actual basic mechanism is vaso-spasm and not vaso-dilation. An understanding of its *modus operandi* via the sympathetics is grasped readily as the sympathetics are the vasoconstrictors.

CLINICAL DISCUSSION.

We must recognize five clinical conditions that will produce otorhinologic headache.

I. Sinusitis:

Nearly every case of headache coming to us, comes self-diagnosed as sinus headache or simply as "sinus condition," a term as vague to me as to the patient who uses it. Although the present swing of the pendulum leads us away from too frequent diagnosis of sinusitis we must not lose sight of the fact of its existence. While it is not the most frequent cause of otorhinologic headache, it does occur with fair frequency; the headache that it produces may be very severe and may be the indication of serious illness.

There is a significant diagnostic difference between the location of the pain and the location of the tenderness in headache of sinus origin. The location of the pain may be very deceptive. For example in our own experience frontal headache is caused by maxillary sinusitis far more frequently than by frontal sinusitis. The location of tenderness, however, is of marked diagnostic value; thus tenderness at Ewing's point on the floor of the sinus at the inner upper angle of the brow almost invariably means frontal sinusitis.

The cause of the pain may be a blocked sinus ostium with increased positive or negative pressure within the sinus. Based on theoretic considerations it has been said that vacuum sinus headache is impossible of existence. One has only to insert a needle into a maxillary sinus that is under negative pressure and literally to see the pain vanish with the inrush of air, to banish such a misconception forever.

McAuliffe, Goodell and Wolf[3] have shown the site of sinus pain to be in the inflamed and edematous duct or ostium and in the inflamed and swollen areas immediately surrounding the sinus opening on both its intrasinal and its intranasal sides. While the *cure* of sinus pain obviously rests on ablation of the underlying infection and so is outside the purview of this discussion, much *relief* may be secured by shrinkage *and anesthesia* of the area in and about the ostium or duct—the source of the pain impulses.

II. Neuritis:

It is in the field of otorhinologic neuritis that most failures to find the cause and to render relief for otorhinologic headache occur.

In general there are three varieties of neuritis of otorhinologic concern:

The first of these is the result of pressure contacts within the nose. These contacts occur between the middle turbinate and the septum or the lateral nasal wall. The headache of this origin varies in distribution with the location of the contact. In general it may be said that the farther forward the contact the farther forward the pain; the farther posterior the contact, the farther posterior the pain.

The second variety of neuritic headache is the "syndrome of the nasal (spheno-palatine) ganglion neurosis" first described by Sluder in 1908 and 1910.[4,5,6] This is a very real and a frequently present clinical entity, and failure to recognize it constitutes a grave clinical error.

The symptoms of this disturbance are widespread, and we will presently understand the reason for this. Pain may radiate to the head, usually keeping below the line of the hat band, and felt most especially in the lower temporal area, the ear, mastoid and occiput. It frequently involves the nape of the neck, the side of the neck and even the shoulder and arm. It may extend to the trunk or leg.

There may be accompanying lacrimation with itching or pain of the pharynx or palate. True cricoidynia, tightness of the throat, especially on swallowing, and even dysphagia are quite common.

In the presence of this disturbance there is almost always tenderness at Sluder's point on the mastoid posterior to the external auditory meatus. I once stood helplessly by as a general surgeon opened a normal mastoid to relieve this symptom which was screaming its own diagnosis.

Sluder relieved these symptoms by intranasal topical or injection anesthesia of the spheno-palatine ganglion area. On the basis of the neurological concept of the time that there were synapses of both the sensory seventh fibers and the sympathetic fibers from the carotid plexus in this ganglion, Sluder attempted to explain the entire process of the symptoms and their relief. As the prime cause of the neuritis he postulated infection, active or latent, in the sphenoid sinus or the posterior ethmoid cells, because of the close anatomical relation of the Vidian nerve to these structures.[7]

As soon as it was shown that all these synapses were not present in the ganglion and that spheno-ethmoidal infection was not always present, Sluder's entire work (both his observed facts and his supporting theories) was attacked and, unfortunately, discredited. This is quite understandable as all of us feel insecure in the presence of a clinical observation that does not seem to rest on accepted anatomic and physiologic bases.

Today with a better understanding of the mechanism of the autonomic nervous system we are on firmer ground, and Sluder's work must be re-evaluated.

On the central side of the ganglion are the sympathetic, the parasympathetic and the sensory supply (see Fig. 1.).

The sympathetic supply consists of non-synapsing postganglionic adrenergic (vasoconstrictor), cholinergic and (most probably) sensory fibers from the cervical sympathetic ganglia via the carotid plexus and the deep petrosal nerve and the Vidian nerve to and through the sphenopalatine ganglion.

The parasympathetics consist of secretory and vasodilator fibers which arise in the nucleus of the VIIth nerve and which reach the ganglion via the great superficial petrosal nerve.

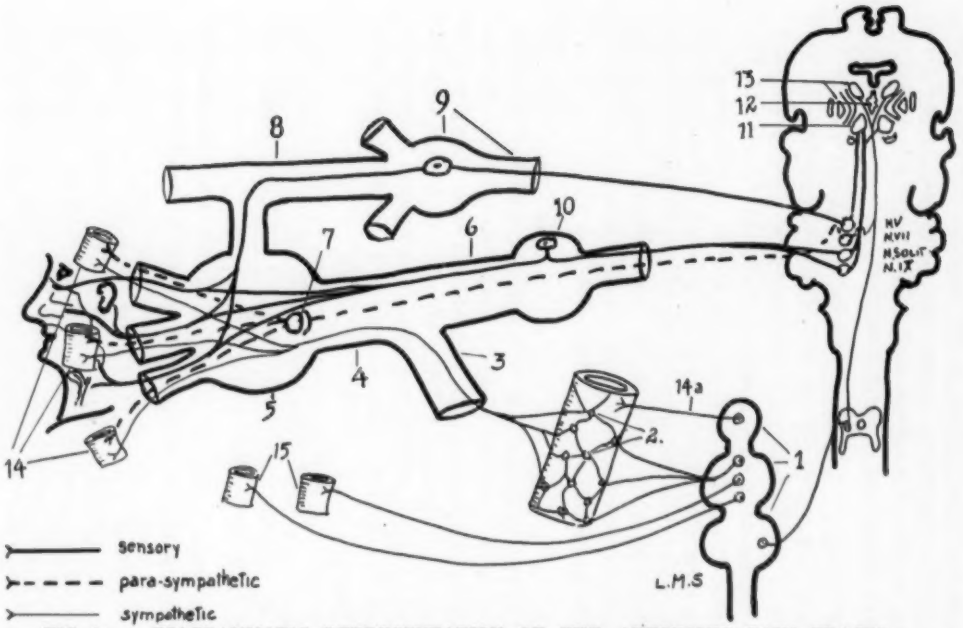

FIG. 1: A DIAGRAMMATIC REPRESENTATION OF THE AUTONOMIC PAIN TRACTS.
(The structures are numbered in the order of their mention in the text.) 1. The Cervical Sympathetic Ganglia; 2. The Carotid Plexus; 3. The Deep Petrosal Nerve; 4. The Vidian Nerve; 5. The Sphenopalatine Ganglion; 6. The Great Superficial Petrosal Nerve; 7. A Parasympathetic Synapse Within the Sphenopalatine Ganglion; 8. The Maxillary Division of the Vth Nerve; 9. Semilunar Ganglion and Trunk of the Vth Nerve; 10. The Geniculate Ganglion; 11. The Thalamus; 12. The Hypothalamus; 13. The Basal Ganglia; 14. The Vessels of the Head, Face and Neck (Including The Carotid); 14a. Auxiliary Fibers Direct to the Carotid; 15. Vessels of the Limbs and Trunk.

These fibers apparently are the only ones having synapses within the ganglion.

The sensory fibers coming from the periphery travel centrally through the ganglion without synapsing there and then, either via the maxillary division of the Vth nerve to the semilunar ganglion, or via the great superficial petrosal nerve and the geniculate ganglion (the so-called sensory VIIth nerve), to the nucleus solitarius and the nucleus of the IXth nerve.

The peripheral origins and distribution of these nerves leading to and from the ganglion on its peripheral side include the orbit, the ethmoid and sphenoid sinuses, the nasal cavity (both septum and turbinates), the nasopharynx, the hard and soft palates, the tonsil and finally the larynx (via the IXth nerve fibers of the sensory VIIth).

We now find that we are in a position to understand much that Sluder could not understand.

There are untold numbers of reflex arc fibers, either uninterrupted or synapsing in the ganglion, which pass through that structure. It is a great crossroad of major trunk lines as well as a lesser relay station. Reflex impulses pass centrally over the fibers of the fifth and the sensory seventh nerves to the thalamus and the hypothalamus, and efferently from the cerebral cortex via the pyramidal system and from the lower motor centers of the basal ganglia to the motor seventh nucleus and to the thoraco-lumbar sympathetic system—most especially to the superior, the middle, the stellate and the lower cervical ganglia. From these latter ganglia impulses travel via the sympathetic fibers of the carotid plexus, through the sphenopalatine ganglion to the vasa vasorum of the arteries of the face, head and neck, or directly from the cervical ganglia to limb and trunk arteries. It is these impulses, vasoconstrictor in type, which produce Hilger's type of vascular pain —the pain perceived in this syndrome.

A frequent accompaniment of this headache is myalgia of the sternomastoid or the trapezius muscle which is produced by a localized round cell infiltration, probably surrounding a vessel whose vasa vasorum are in spasm. In these cases the pain is referred to the insertion of the involved muscle—that

is to either the mastoid process or to the occiput, whereas the tenderness is in the belly of the muscle involved at the site of the infiltration. When the sternomastoid is involved the localized tender infiltration must not be mistaken for the true carotid pain of Hilger. Relief is afforded by heat and gentle massage along and in the direction of the muscle fibers.

Sluder's syndrome may be accompanied by vasomotor labyrinthitis. This will be discussed later with psychogenic or tensive headache. Here in contrast to psychogenic headache and vertigo there is a local anatomic reason for the pain as well as the vertigo.

The physical causes for sphenopalatine ganglion neuritis are a contact between a far posterior septal spur with the posterior tip of the middle turbinate; inflammation of the posterior group of sinuses, and very frequently, because of a marked concavity of the septum or an atrophy or absence of the posterior tip of the middle turbinate, the sphenopalatine ganglion area is exposed to an inspired blast of irritating raw air.

The diagnosis of any of these headaches of neuritic origin lies in the repeated relief of the pain by anesthesia of the points of contact or the terminal filaments of the nerves involved or the ganglion area.

The treatment is indicated immediately by the diagnostic procedure. This treatment consists of interruption of the afferent impulses from the contact pressure or from the irritated ganglion. Repeated anesthesia of the contact or ganglion area may in itself be effective without any further procedure. Infraction or exfraction of the offending turbinate or its electrocoagulation may be required. Submucous resection of the nasal septum is a frequent requirement. The resection should be complete, not only to remove the contact or to correct a faulty breathway, but also to act as a decompression procedure, so that when the turbinates later engorge physiologically they will not impact against a rigid and unyielding wall.

Frequently after surgery has been performed sessions of

anesthesia may have to be continued for a short time if the pain is not relieved completely by the operation. Here a "habit pain pattern" has been set up in the reflex arc that persists after the removal of the physical irritant. It is the interruption of this arc by anesthesia that finally effects the cure. When topical anesthesia fails to relieve the pain, blocking of the stellate ganglion area by the method of Alexander[8] should be done. This is highly effective as the blocking agent in this particular procedure reaches the lowest sympathetic fibers, although the accompanying Horner's syndrome may be disturbing to an introspective patient.

The third and last neuritic type of headache seemingly has not been described previously although it has been hinted at under such terms as "atypical neuralgia" or "atypical face pain." I have not worked out definitely the tracts involved, but I feel that this headache is based on the conception of White, Smithwick and Simeone that there are pain tracts in the sympathetics.[9] For this reason I have spoken of it as sympathetic neuritis. Eventually it may be demonstrated to be only a variation of vascular pain.

It is characterized clinically by a peculiarly indescribable nagging pain, usually starting in or about a tooth that evinces no apparent pathology. The tooth usually is extracted against the better judgment of the dentist, because of the insistence of the patient who is terribly annoyed by the pain. If the dentist holds out against the extraction, and the patient endures the pain which no treatment seems to relieve, the pain usually disappears; but if the tooth is extracted, mischief is afoot: the pain increases; then follows extraction of one of the abutting teeth or perhaps of both of them. This is followed in turn by repeated curetment of the socket and biting away of the alveolar ridge.

Blessed is the patient if this occurs in the lower jaw because no sinus abuts it. If, unfortunately, it is an upper tooth the antrum is next attacked; then the ethmoids, and finally the sphenoid. With each succeeding surgical procedure the pain becomes distressingly worse. I know this, because I performed the last three of a series of operations suffered by the patient in the first case that I ever saw and recognized.

I reoperated a foully infected antral pocket after the victim had had all molars and premolars on the affected side extracted and then had suffered a combined radical antrum and ethmoid exenteration. Later I performed a sub-mucous resection and finally evulsed the infra-orbital nerve which resulted in complete second division anesthesia in the affected area but without relief of the pain. It was after the last procedure that it finally dawned on me that the pain was following the blood vessels and not the second division of the Vth nerve.

With this recognition of a clinical entity other cases soon appeared in the practice. It is not a frequently met situation, but if it can be recognized, preferably before the first dental or surgical attack, the patient and the doctor will be happier for it. It does seem to be self-limited if only the patient can be prevailed upon to leave it alone; otherwise I know of no treatment for it.

III. Psychogenic or Tensive Headache:

This headache is the twin sister of psychogenic vasomotor labyrinthitis which produces vertigo and hearing disturbances, and they frequently appear together.[10] It operates via the same mechanism as sphenopalatine ganglion neuritis, following the same anatomic vicious circle, but its point of origin lies not in the ganglion or in the periphery but in the diencephalic structures—notably the thalamus and the hypothalamus. Here the cure rests on two factors. If possible the psychic cause should be discovered and made clear to the patient *by the otolaryngologist* if at all possible, at whatever cost of time and effort to him; the patient's cooperation should be obtained in rearranging his work, worry and eager-beavering, and in altering his responses to his problems of life. Pharmacologic interruption of the vicious circle by anesthesia of the ganglion area is the second and equally important factor in effecting the cure. Again it is the mechanical or pharmacological breaking of a "pain habit pattern" that may be necessary for a cure. Either factor without the other may prove to be of little avail.

IV. Histaminic Cephalalgia:

This headache first described by Horton[11] is a definite clinical entity which must be recognized by the otolaryngologist in his consideration of headache, as it may well be mistaken for neuritic headache. To enter into a discussion of a subject covered so well by Horton in his various publications is unnecessary. Suffice it to say that this headache is met frequently, and its symptoms are so severe as to be disabling. When correctly diagnosed and treated, the results are so dramatically satisfactory that it sires a patient whose gratitude knows no bounds.

V. Neoplasms:

These offer such a wide range of pain symptoms that a monograph would be required to describe them; but three facts must be mentioned:

Tumors of the fossa of Rosenmueller—usually epidermoid carcinoma, transitional cell type—may produce typical Sluder's syndrome. Let us always examine this region for such a possibility in the presence of the syndrome of sphenopalatine ganglion neuritis.

Tumors of the larynx may have as their first symptom severe otalgia, and unexplained ear pain calls for meticulous laryngoscopy.

Finally, cases of malignancy of the nasopharynx that have been treated with radiation therapy often develop severe unilateral headache after most of the reaction has subsided. For some as yet unexplained reason anesthesia of the ganglion area seems to be the most effective means of relief.

CONCLUSION.

We find that another expanding field has been reopened to otolaryngology. The physician who can relieve what may well be the most widespread and most frequent complaint of modern man is in an enviable postion. Our strategic position in the reawakened field of otolaryngology where we are apt to be

the first physician to see the patient with headache and where we are the best equipped of all branches of medicine to understand and to conquer the problem, makes us doubly to be envied.

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TREATMENT OF ACUTE SINUSITIS WITH PENICILLIN AND THE INTRAMUSCULAR USE OF STREPTOKINASE.*

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Acute sinusitis is a diffuse inflammation of the mucosa of the sinuses with hyperemia, edema and serous exudation. The edema produces occlusion of the ostium of the sinus, the normal route for escape of secretions. A favorable therapeutic response will be obtained in many patients treated early in the course of the disease with a combination of chemotherapeutic agents, antibiotics, local application of vasoconstrictor drugs and sedation for relief of pain. Progression will be seen in some patients in spite of such measures. The ostium will remain blocked, the edema will increase in amount, and pus will collect. If drainage is not provided, subperiosteal abscess, osteomyelitis, meningitis, extradural abscess, intracerebral abscess or chronic sinusitis may occur. The patient who fails to respond to conservative measures must be treated by surgical drainage of the sinus to prevent complications.

Recently it has been shown that the intramuscular injection of streptokinase will reverse the inflammatory state and reduce edema.[1] The morphologic changes of inflammation seen

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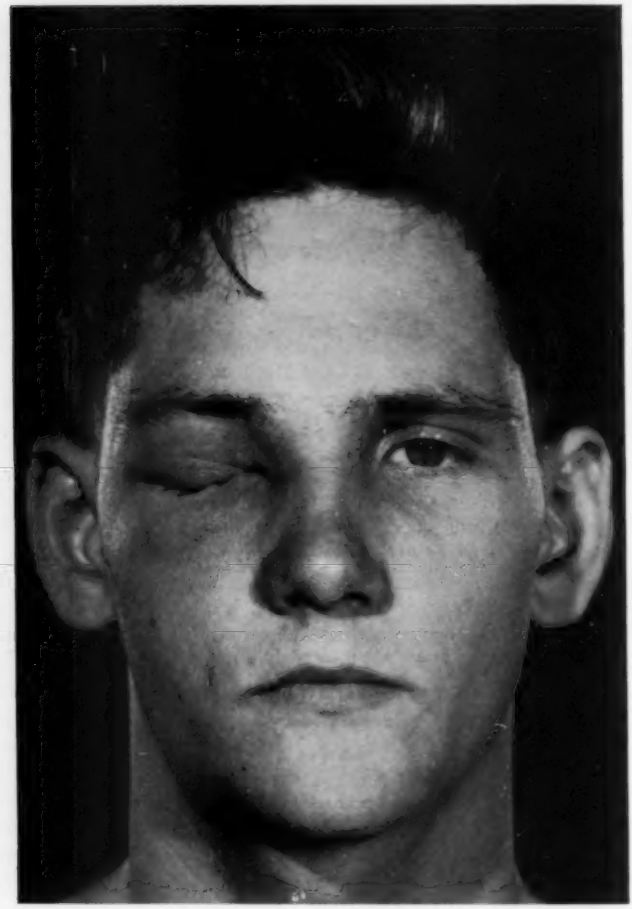

Fig. 1. Appearance of the face on September 17, 1954.

in the tissues are the mechanism by which an injurious agent is localized in the body. These barriers not only prevent spread of infection, but also stop the adequate delivery of anti-bacterial substances from the rest of the body to the infected area. Streptokinase will remove some of these hindrances so that greater amounts of anti-microbial drugs can

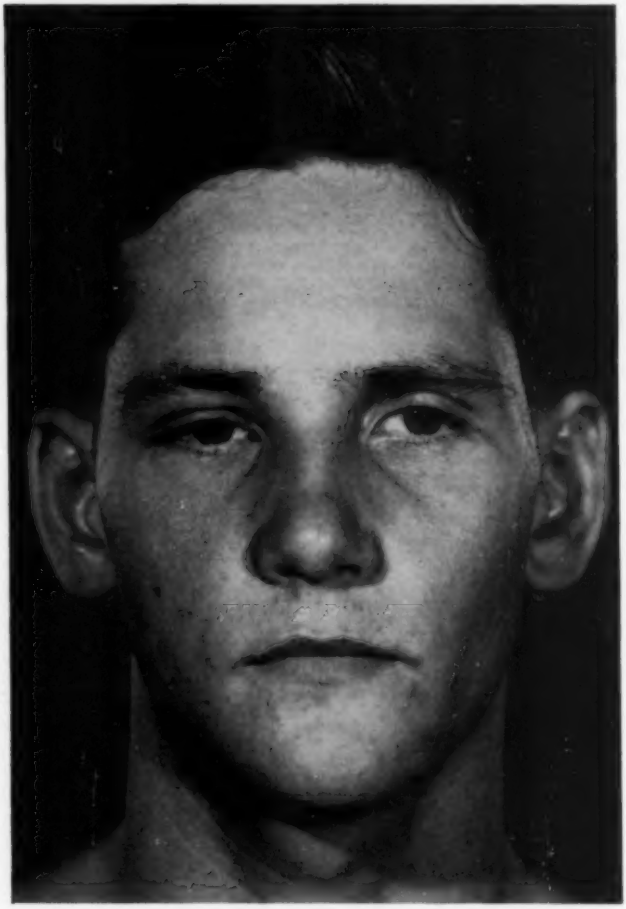

Fig. 2. Appearance of the face on September 20, 1954.

go to the involved site. The intramuscular injection of streptokinase cannot be used without the concurrent administration of anti-bacterial agents, or spread of the infection might be facilitated.

Varidase, composed of streptokinase and streptodornase, was dissolved in physiologic saline so that the final concen-

tration of streptokinase was 10,000 units per cc. The solution was stored in the refrigerator when not being used. A fresh solution was made daily. The effects achieved by the injection of varidase were ascribed to streptokinase, since the observed therapeutic effects were not produced by the intramuscular administration of a purified preparation of streptodornase.

Case Report—A 20-year-old white man was admitted to the hospital on Sept. 16, 1954, with an acute right frontal sinusitis of five days' duration, associated with periorbital edema and occlusion of the palpebral fissure. Severe headaches, chills and fever had been present. The patient had received three injections of an unknown amount of penicillin by his family physician without effect.

The temperature was 101.4°F. There was redness and edema of the right periorbital area (see Fig. 1), and tenderness over the frontal sinus. The mucous membrane over the right middle turbinate was red and swollen. Roentgenograms showed clouding of the right frontal and ethmoid sinuses.

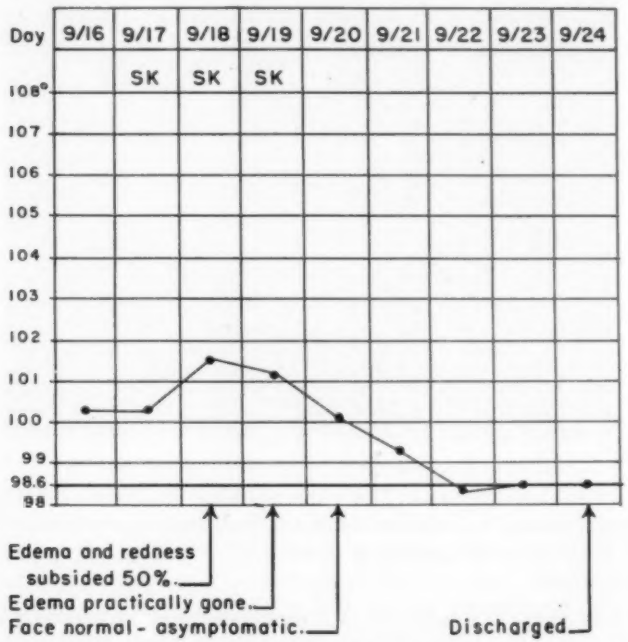

Fig. 3. Clinical course of the patient.

Packing, saturated with solutions of 10 per cent cocaine hydrochloride and 1:1000 epinephrine, was put into the right side of the nose, four times daily, and 20 drops of 0.25 per cent solution of neosynephrine were placed into the same area every three hours from Sept. 16 through Sept. 20. The patient was given 300,000 units of procaine penicillin G in aqueous solution and 0.5 gram streptomycin sulfate, both intramuscularly, twice daily from Sept. 16 through Sept. 24. Intramuscular injections of 5,000 units of streptokinase in 0.5 cc. of physiologic saline were given twice a day from Sept. 17 through Sept. 19. On Sept. 18, the periorbital edema had started to subside (see Fig. 2), and the headache had disappeared. By Sept. 19 the patient was asymptomatic, and the edema was practically gone (see Fig. 3). On Sept. 20 all evidence of infection had disappeared. The patient was afebrile on Sept. 22. The patient was discharged from the hospital on Sept. 25.

SUMMARY.

Streptokinase administered intramuscularly with the antibacterial drugs was a valuable adjunct in the treatment of a patient with an acute right frontal sinusitis. It is suggested that, because of the beneficial effects observed in this patient, this method can be used in the office management of patients, whose disease is not quite so severe, to prevent hospitalization. Further investigation of this treatment is indicated.

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THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY CONVENTION CRUISE

The Virginia Society of Ophthalmology and Otolaryngology is sponsoring a convention cruise to Havana and Nassau on May 26 to June 2, 1956. Sailing from and returning to Norfolk, Virginia, the "Queen of Bermuda" will act as the hotel for the trip. Fare for seven days, $165.00 and up per person. Make reservations with United States Travel Agency, Inc., Washington, D. C.

CONGENITAL ATRESIA OF THE EXTERNAL AUDITORY MEATUS.*

MERRILL GOODMAN, M.D.,

New York, N. Y.

Surgical correction of congenital atresia of the external auditory canal has been attempted many times over the years with very little success before 1947, when Pattee[1] reported on five cases and showed good results in four of them. Prior to that time Fraser[2] and Richards[3] had reviewed the embryological and anatomical anomalies that are generally found.

The inner, middle and external ears develop from different anlages and, therefore, congenital malformations of the canal are rarely associated with inner ear changes. The inner ear develops first from an invagination of the ectoderm that forms the otocyst from which the cochlea and labyrinth develop.

The external ear and Eustachian tube develop from the first pharyngeal pouch. Simultaneously, there is an outpocketing of the entoderm to form the Eustachian tube and a pitting of the ectoderm at the site of the future external auditory meatus. A cord of cells is formed from the ectoderm to the entoderm, where a plate forms just lateral to the middle ear cavity. This cord of cells later canalizes from within out, leaving the tympanic membrane at the site of the junction of the ectoderm and entoderm. The auricle forms from six mounds developed from the first and second branchial arches around the first branchial groove. The malleus and incus develop from Meckel's cartilage of the first branchial arch, and the stapes arises from Reichert's cartilage of the second branchial arch.

The os tympanum develops directly from membranous bone as a U-shaped bone surrounding the ectodermal core previously described, and it subsequently forms the sides and floors of the bony external auditory canal.

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* Read before the New York Academy of Medicine, Section of Otolaryngology, May 18, 1955.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 6, 1955.

Failure of the ectodermal core to canalize results in a deformed tympanic bone, and an overgrowth of bone occurs to fill most of the space normally occupied by the external auditory meatus.

Associated with congenital atresia of the canal are varying degrees of deformity of the auricle and the incus and malleus. There are also varying degrees of formation of the tympanic cavity, and an associated pneumatization of the mastoid bone.

The surgery of choice depends upon the extent of the malformation within the tympanic cavity. Pattee believed that previous failures to correct the hearing were due to neglecting the fact that the incus and malleus were usually deformed and that they were probably fused into position, thereby immobilizing the stapes; therefore, he removed the incus and checked the motion of the stapes in each case before applying a Thiersch graft to form a pseudo-tympanic cavity.

In 1947, also, Ombredanne[4] described two cases in which he did a similar operation and a fenestration in one stage with excellent results in one case. He later reported[5], in 1952, on 33 cases done with the same one-stage technique.

Meanwhile, in this country, several authors including Altmann[6] suggested the possibility of doing a fenestration in one or two stages for the same deformity. Woodman[7] in 1952 reported a case in which he first operated using the Pattee method but carefully skeletonized the horizontal semicircular canal. At a later date, following little or no improvement in the hearing, it was a relatively simple procedure to raise a flap and do a fenestration with excellent functional result.

On the service of Dr. Ward C. Denison at the New York Eye and Ear Infirmary, we recently operated on a seven-year-old boy with a complete atresia of one ear and an almost complete occlusion of the other. We are presenting his case, further to emphasize the fact that in congenital anomalies any combination of malformations can occur, and each case must be individualized.

Case Report: (H. H.) This seven-year-old white male was sent home from school with a note stating that the teacher believed that his difficulties in school were caused by his inability to hear properly. He had no complaints of pain at any time, and only by careful questioning were

we able to elicit the fact that he occasionally had drainage from the left ear. His past medical history revealed no previous illnesses. His tonsils and adenoids had been removed at the age of four.

Physical examination revealed a well nourished, well developed boy with abnormalities limited only to the ears. Both auricles were perfectly formed and properly placed. The right ear showed a shallow depression leading to a blind pouch in place of a normal external auditory meatus. In the left ear there was extreme narrowing of the meatus to approximately 1 mm. in diameter, allowing no view of the tympanic membrane.

Radiographs of the temporal bones revealed no discernible evidence of the external auditory meatus with partial pneumatization of each mastoid.

Audiogram showed an average 45 db loss in the speech range of the right ear, and a 35 db loss in the speech range of the left ear. Because the right ear had the poorer hearing, surgical intervention of that side was proposed.

On June 16, 1954, under general anesthesia, a right endaural incision was made, and the mastoid bone was exposed. The cortex overlying the antrum was removed. The periantral cells were exenterated and the horizontal semicircular canal was skeletonized. Using the canal as a landmark, the zygomatic arch, posterior and superior canal walls and the bone filling the area of a normal external auditory meatus were removed with the drill and cutting burrs.

The boy was found to have a complete soft tissue atresia, and the skin was excised anteriorly to what seemed the normal anterior margin of an external auditory meatus. The bony occlusion was of an hour-glass shape, and did not continue down to the tympanic membrane. The membrane itself was in an infantile (or more horizontal) position, and was not of a normal color or consistency. It contained a large perforation. The ossicles appeared to be normal and were left in place. The entire cavity was then lined with a split thickness graft taken from the thigh, and was packed with cotton pledgets.

The right ear healed quickly, and in less than two months there was no further discharge, and the hearing had improved to an average of 20 db below normal, an increase of 25 db.

On Sept. 15, 1954, the left ear was operated on in a similar manner. On the left side there was not a complete occlusion of the canal, but rather only a marked stenosis. Cholesteatoma was found in the antrum and attic with a large tympanic membrane perforation. All mastoid air cells were carefully exenterated, and the ossicles were cleaned but left in place. Again a split thickness graft was applied.

Recovery on the left side was slower than that of the right, because of cholesteatoma and infection; however, in four months the entire cavity was healed with dry perforations of the tympanic membrane.

The last audiogram, taken on Feb. 12, 1955, showed an average loss of 15 db in the speech frequencies of each ear. This amounts to a gain of 30 db in the right ear and 20 db in the left ear. This boy has improved in school to such an extent that he has been advanced to the stage of his normal

age group, from which he was dropped one year ago. He is happy, alert and responsive, and has shown a marked change from his phlegmatic personality prior to surgery.

SUMMARY AND CONCLUSIONS.

A brief survey of the embryological factors involved and history of the surgery for congenital atresia of the ears is given. A case report illustrationg an excellent functional result in a boy with complete atresia of one ear and partial occlusion of the other, complicated by cholesteatoma, is discussed.

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DACRYOCYSTORHINOSTOMY: SIMPLIFIED PROCEDURE.*

HENRY M. GOODYEAR, M.D.,

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Numerous operative procedures have been advanced over the years for the relief of chronic dacryocystitis, mucocele and epiphora; in fact, it would be difficult to offer any operation which at least has not been described as a part of some often rather extensive or complicated procedure. The many operations described only emphasize the lack of a generally accepted procedure.

From the many operations offered and from a varied personal experience over the past thirty-five years, in which I have had a particular interest in this work, I am convinced that the operation can be done simply, without suture of the lacrimal sac to the nasal mucous membrane or without transplanting any part of the sac into the nose or extending any material such as tubes, sutures, or packing into the nasal passage.

INDICATIONS.

Operation is indicated in any chronic dacryocystitis or chronic epiphora where the inferior canaliculus is open as is indicated by a return of tear or pus into the eye when pressure is made over the lacrimal sac. The obstruction is usually at the mouth of the lacrimal duct where it often narrows as it leaves the lower portion of the sac. Age: my patients have ranged from 4 to 78 years.

SURGICAL ANATOMY.

The lacrimal sac (see Fig. 1) is on the average 12 mm. long[1] and 2 to 5 mm. wide, and the dome is usually from 2 to 5 mm. above the exit of the orifice of the canaliculi; thus, it is evident that the maximum opening of only 1 cm. can be

* Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, in Cincinnati, Ohio, January 16, 1956.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, January 16, 1956.

made in the vertical direction of the medial surface of the sac at operation. With a maximum width of only 2 to 5 mm. it is true that any opening much wider than 7 mm. in the bone of the lacrimal fossa into the nose is of little importance.

Usually only very thin lacrimal bone (see Fig. 2) (often of paper thickness) and the posterior edge of the frontal process of the maxilla bone form the medial bony wall of the lacrimal fossa (approximately half and half). Very commonly, however, according to Schaeffer[1] two or more anterior ethmoid cells (frontal and infundibular) immediately adjoin the dorsal and medial aspect of the lacrimal sac; even a dehiscence may be present. He states that "even more common is the extension of anterior ethmoid cells over the medial or nasal side of the lacrimal sac and the beginning of the nasolacrimal duct." The rhinological problem thus becomes more important in dacryocystorhinostomy than the ophthalmological problem of dealing with the lacrimal sac. The entire wall of the lacrimal fossa may be pneumatized by ethmoid cells which must be eliminated at operation.

Another rhinological problem of importance is that of a deviation of the nasal septum which must be corrected at the time or preceding the dacryocystorhinostomy.

The anterior lacrimal crest is of the maxillary bone while the posterior crest is of the lacrimal bone.

OPERATION.

Local anesthesia: Premedication in adults consists of Nembutal or Seconal, grains one and a half, given orally, and morphine, grains 1/6, plus scopolamine, grains 1/150, intramuscularly, forty-five minutes to one hour before operation.

One per cent novocaine containing adrenalin (1:1000) fifteen drops to the ounce is injected along the medial wall of the orbit (1 cm. above the inner canthus) toward the anterior ethmoid nerve. A second injection extends toward the infraorbital foramen and along the line of incision.

Cocain powder and adrenalin is applied to the mucous membrane of the middle meatus.

Incision: A straight line incision is made midway between the midline of the nasal bridge and the inner canthus, as any incision made nearer to the canthus may produce an irregularly drawn scar. The incision extends 1 cm. above the canthus and 2 cm. below.

Frequently the inexperienced operator carries the incision too high, even into or below the eyebrow, because he loses sight of the fact that the sac lies almost entirely below the canthus. So high an incision results in unnecessary bleeding and an awkward approach to the sac.

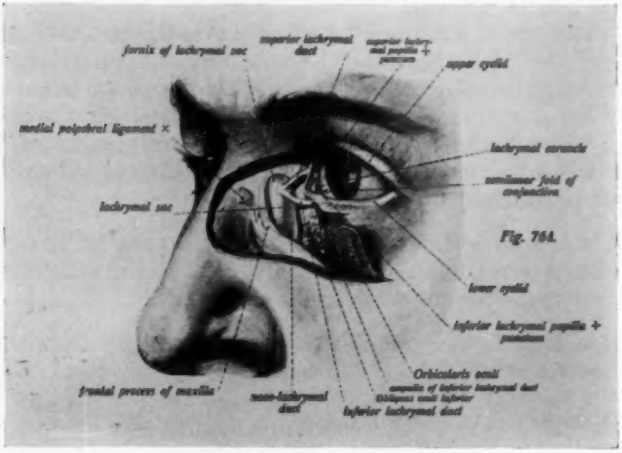

Fig. 1. Lateral wall of the lacrimal sac. (From Atlas and Text Book of Human Anatomy, p. 269, by Sobotta McMurrich. Saunders and Co.)

The incision is carried immediately through the soft tissues and periosteum to the bone.

The soft tissues and periosteum are elevated, together with the medial palpebral ligament which is not cut. The elevation is carried medially by means of a fairly sharp submucous elevator, starting first over the frontal bone above the lacrimal fossa exposing the upper end of the anterior lacrimal crest and the upper portion of the fossa. The posterior crest is exposed, but the sac is not elevated beyond this point. As the

elevator is moved downward into the fossa the nasal wall of the sac is easily exposed well into the mouth of the lacrimal duct.

The attachment of the lateral wall (see Fig. 1) is not disturbed, as this attachment will serve to keep the sac in a better position post-operatively.

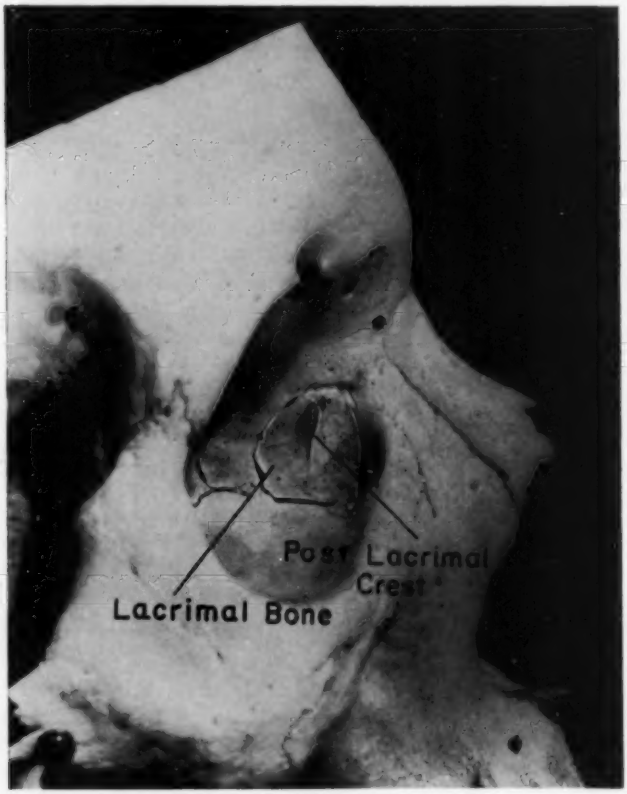

Fig. 2. Lacrimal bone, showing its posterior lacrimal crest.

Bone fenestration: The lacrimal bone (see Fig. 2) is often of paper thickness and can easily be opened by means of a

sharp 2 or 3 mm. mastoid curette (Spratt) held firmly in one hand while being turned medially and then outwardly with the opposite hand. All the bone necessary may be removed by means of the curette without opening the nasal mucous membrane. The bony opening is extended well into the mouth of the lacrimal duct and this extension is important. This opening is also extended to the posterior lacrimal crest and including the crest, especially in its lower portion. The bone is removed upward to the level of the dome of the sac.

The medial edge of the anterior lacrimal crest may be shaved away for 1 to 2 mm. well down into the mouth of the duct. I do not attempt to remove heavy thick bone of the maxillary process which adds only to the formation of granulations in healing.

The mucous membrane of the nose, if not already opened, is incised and cut away cleanly just to the size of the opening previously made in the bone; thus, the nasal mucosa over the lacrimal sac is sacrificed completely. A Hartman middle ear, through-and-through biting forceps is very helpful in the final trimming of the soft tissues, as the edge must be cut clean and not dissected up beyond the edge of the bony opening. If anterior ethmoid cells intervene, a second bony wall is dealt with.

The medial wall of the lacrimal sac is lifted by means of a dural hook and opened in its midline with a small knife. The incision is carried in the vertical plane well into the mouth of the lacrimal duct and upward within 2 mm. of the dome of the sac (see Fig. 3). A horizontal incision is then made the full width of the sac at the upper end of the vertical incision, 2 to 3 mm. from the top of the dome, which will expose the mouth of the common duct of the canaliculi.

A second horizontal incision is made at the lower end of the vertical incision as far into the mouth of the duct as possible as it is in this area that the opening into the nose can be established well below the anterior end of the middle turbinate. Here one has to deal only with thin bone and nasal mucosa.

The incisions in the nasal wall of the sac having been carefully and widely procured, the long posterior flap of the

sac is laid into the nasal opening. The anterior flap is turned anteriorly and held in position by means of two 4-0 plain catgut sutures carried into adjacent soft tissues on the medial side. I have, however, laid this flap into the nasal opening without suturing, with excellent results; once the sac is opened widely there seems to be no tendency for the sac to reform. It is the nasal opening which offers our chief problem.

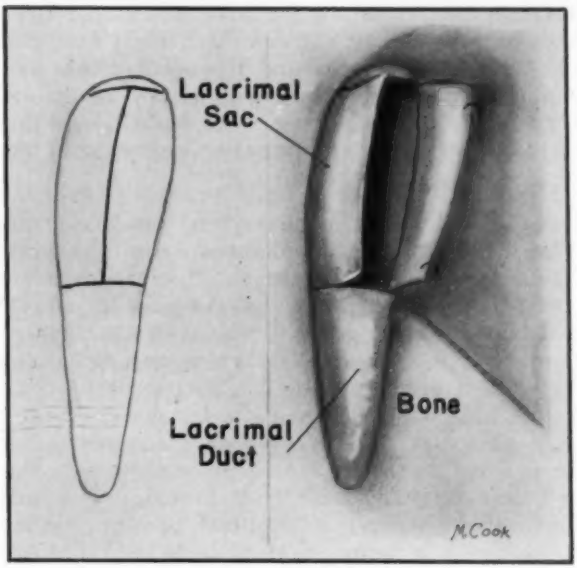

Fig. 3. Diagrams of incisions made in the nasal wall of the lacrimal sac.

The soft tissues are laid back into position and the skin closed by two figure-eight fine silk sutures which are removed on the fourth day; often cut on the second day.

Vaseline ointment is applied to the eye and a light bandage applied for twenty-four hours.

No packing is placed in the nose, and the patient leaves the hospital on the day after operation.

After Care: The patient is up on the first day. The sutures are removed by the fourth day, sixth in children, and the nose examined for blood clots.

Usually tearing stops immediately, and by the seventh day gentle pressure may be applied intermittently by the patient to milk the sac as a guard against a retarded flow of tears into the nose. A 5 per cent solution of silver nitrate may be applied at the end of a week or ten days in the middle meatus. Should some tearing occur a probe is passed through the inferior canaliculus and the distal end moved upward in the nose to separate any adhesions that might have occurred. Should this procedure fail and the opening into the nose close, the following procedure will usually re-establish the nasal opening. It is a scarring of the nasal tissues that produces the closure—not a reformation or closing of the wall of the lacrimal sac.

The procedure of reopening the tract consists of injecting the area of the sac with 1 per cent novocain and cocainizing the nose. A probe is passed through the inferior canaliculus and pressed firmly against the inner wall of the nose. While an assistant holds the probe firmly in this position a right angle tonsil knife with a short blade is used to cut down on the probe as it presents in the middle meatus. A triangular incision is made with the base of the triangle downward. The probe is passed at five to seven day intervals for several weeks and the distal end moved up and down in the nose to prevent a new membrane from forming. The patient is also instructed to apply intermittent pressure against the sac to produce a pumping or intermittent suction effect.

In most cases no after treatment is required, especially where the nasal mucosa has not been unduly traumatized beyond the opening made in the bone. None of my cases have been complicated by hemorrhage, and often no treatment whatever has been applied to the nose following operation.

SUMMARY.

I have depicted in a practical way the surgical anatomy and its importance and limitations in dacryocystorhinostomy.

I have emphasized the wide opening of the lacrimal sac and stressed the importance of its extension well into the mouth of the duct.

I have emphasized the importance of a precise opening into the nose with minimal trauma to the edges of this opening and the complete sacrifice of the nasal mucosa over the area corresponding to the sac.

REFERENCE.

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AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

The Scientific Meeting of the American Society of Facial Plastic Surgery will be held Wednesday evening, March 7, 1956, at the Biltmore Hotel, New York City. Program will be as follows:

I. Septal Surgery in Rhinoplasty (Rapid Procedure). Dr. Jesse Fuchs—Member Attending Staff, Cedars of Lebanon Hospital and Mount Sinai Hospital, Los Angeles, California.

II. Otoplasty for Protruding Ears (Movie Presentation of Technique). Dr. David Meyers—Professor and Head of Department of Otolaryngology, Temple University, Philadelphia, Pennsylvania.

DISCUSSION. Dr. I. B. Goldman — Rhinoplastic Surgeon, Mount Sinai Hospital, New York City.

III. Use of Thorazene in Anesthesia and Its Application to Rhinoplastic Surgery. Dr. LeRoy W. Krumperman—Professor of Anesthesiology, Temple University Medical Center.

IV. Jacques Joseph's Last Rhinoplasty — Outcome. Dr. Gustave Fred—Consultant Otolaryngology, Beth Israel Hospital and Massachusetts General Hospital, Boston, Mass.

Make reservations for yourself and guests at your earliest convenience. Subscription $15.00 per person, all inclusive. William Schwartz, M.D., Secretary, 224 Lexington Avenue, Passaic, N. J.

BOOK REVIEW.

Bronchography in Children (La Broncografia nel bambino). By Lucio Parenzan and Alfredo Vago. Supplement XXIII of the Archivio Italiano di Otologia, Rinologia e Laringologia, 1955 (In Italian. Summaries in English, French and German).

This is an authoritative contribution to the knowledge of lower respiratory pathology in children.

A new and safe technique of bronchography under general anesthesia is described with excellent results obtained in over one hundred children. Several examples of normal anatomy and tracheobronchial topography are presented. The authors list the indications for bronchography in children as tracheobronchial pulmonary malformations, chronic bronchopulmonary suppuration and primary tuberculosis.

Bronchography means team work between bronchoscopist, radiologist and anesthetist. It becomes the essential item of a triad of diagnostic procedures in lower respiratory pathology: laminography, bronchography and bronchoscopy. The authors suggest that in children even bronchoscopy should be "guided" by bronchography.

The exposition is clear and simple, and many references are given. The monograph is amply documented by outstanding illustrations with explanatory diagrams making the reading extremely easy and enjoyable.

E. J. F.

ERRATA.

The following legends were inadvertently omitted from the article on Acetylcholinesterase Activity in the Cochlea, by J. A. Churchill, M.D., H. F. Schuknecht, M.D., and R. Doran, in the January, 1956, issue of The Laryngoscope:

FIGURE 1. Photographs taken through the dissecting microscope.

Upper: Experiment 2. View of treated (T) and control (E) cochleae placed adjacent to each other showing band of copper sulfide precipitate (arrows) in the three turns of the treated cochlea.

Lower: Higher power magnification of the treated cochlea showing the band of copper sulfide precipitate (I) near the margin of the osseous spiral lamina (S).

FIGURE 2. Photographs taken through the dissecting microscope.

Upper: Experiment 1. In the exposed basal turn of the treated cochlea there are bands of copper sulfide precipitate lying in relation to inner hair cells (I) and outer hair cells (O). The osseous spiral lamina is seen at "S."

Lower: Experiment 2. 130x. Teased specimen from basal turn, not counterstained, showing dense band of precipitate (I) related to inner hair cell row and small discrete deposits of precipitates (O) adjacent to the three rows of outer hair cells.

FIGURE 3.

Upper: Experiment 8. Teased preparation from apical turn, incubated in D.F.P., counterstained with acid fuchsin, showing dense band of precipitate (I) lying in relation to inner hair cell row. There is a small amount of precipitate on outer hair cell rows (O). The osseous spiral lamina (S) and spiral ganglion (G) are seen.

Lower: Experiment 6. Microscopic section of organ of Corti from middle turn showing large deposit of precipitate (D) located above the inner phalangeal cell which was detached from inner hair cell (I). There is a small deposit of precipitate in the habenula perforata (H) and at the base of the first outer hair cell (O). The pillars are seen at "P" and the tectorial membrane at "T." It should be remembered that considerable post mortem degeneration is present because about 90 minutes elapsed between death of the animal and fixation of the tissues, 45 minutes of which they were incubated at 38° C.

FIGURE 4. Photograph taken through dissecting microscope.

Upper: Experiment 2. Copper sulfide precipitate in the three turns of treated cochlea. Notice precipitate on nerve fibres (N) in osseous spiral lamina of apical turn.

Lower: Experiment 2. Teased preparation from apical turn showing precipitate on the nerve fibres (N) in the osseous spiral lamina. The organ of Corti and basilar membrane are missing except for a small portion of the inner hair cell row with dense copper sulfide precipitate (I). The small deposits of precipitate (E) along the edge of the osseous spiral lamina appear to be in relation to nerve endings for inner hair cells.

FIGURE 5. Experiment 8. Muscle control. All techniques, including photographic factors, held constant.

Upper: Microscopic section showing deposits of copper sulfide precipitate in the muscle end plates.

Center: Another section from same muscle, treated in addition with D.F.P. to show disappearance of some of the diffuse precipitate believed to be due to non-specific cholinesterase.

Lower: Same, except not treated with substrate acetylthiocholine.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The Sixth International Congress of Otolaryngology will take place in Washington, D. C., from Sunday, May 5, through Friday, May 10, 1957, under the presidency of Arthur W. Proetz, M.D.

The selected subjects for the Plenary (Combined) Sessions to be held Monday, Wednesday and Friday mornings will be:

1. Chronic Suppuration of the Temporal Bone.
2. Collagen Disorders of the Respiratory Tract.
3. Papilloma of the Larynx.

Outstanding internationally recognized authorities will open the discussion of each of these subjects.

Two types of communications are invited: *1.* Contributions to the discussions of the selected subjects, limited to five minutes. *2.* Original papers, limited to 15 minutes. These should be in one of the four official languages: English, French, German, Spanish.

Motion picture films will be shown continuously except during the Plenary Sessions. There will be both scientific and technical exhibits. Those wishing to submit contributions to the program should communicate with the General Secretary.

Announcement of the Congress has been sent to all otolaryngologists whose names and addresses could be obtained. Additional details concerning registration, housing, entertainment, etc., will be sent to those who have indicated to the General Secretary that they wish further information.

The subscription for Members (physicians) is $25.00 U.S.A. This includes the privilege of attendance at all official Congress meetings except the banquet for which an additional charge will be made. Other persons accompanying Members may be registered as Associates at a fee of $10.00 U.S.A.

An interesting program of social functions, visits to points of interest in and around Washington and post-Congress tours is being arranged. The American Express Company is the official travel agent for the Congress. Their offices through-

out the world are available for travel arrangements to the Congress and for post-Congress tours.

All communications should be addressed to the General Secretary, Paul H. Holinger, M.D., 700 N. Michigan Ave., Chicago, Ill., U.S.A.

INDIANA UNIVERSITY MEDICAL CENTER.

The Department of Otolaryngology, Indiana University School of Medicine, offers its annual Anatomical and Clinical Course in Otolaryngology March 26th to April 7th, 1956.

Applicants should address The Post-Graduate Office, Indiana University Medical Center, Indianapolis 7, Indiana.

NEW CLINIC OPENED.

A new Clinic for Reconstructive Plastic Surgery of the Face was opened officially on December 8, 1955, in a new wing of the Manhattan Eye, Ear and Throat Hospital, New York City. Dr. John Marquis Converse is the Surgean-Director of the Clinic.

The staff includes a co-ordinated team of specialists in fields associated with plastic surgery who will evaluate and treat all types of facial disfigurements. A teaching clinic will be developed, and fellowships in plastic surgery are to be made available.

ENDAURAL OTOLOGIC SURGERY COURSE.

A special course in Endaural Otologic Surgery will be given by Northwestern University February 28 through March 26, 1956. Course is limited to eight otolaryngologists. Instruction to include lectures, cadaver dissection, etc. For further information write Dr. Geo. E. Shambaugh, Jr., Dept. of Otolaryn., Northwestern Medical School, 303 East Chicago Ave., Chicago (11), Ill.

NATIONAL SOCIETY MEETINGS.

Schedule of Meetings for 1956:

American Board of Otolaryngology, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 6-11.

American Otological Society, Inc., to be held at the Seigniory Club, Ottawa, Canada, May 11-12.

American Laryngological Association to be held at the Seigniory Club, Ottawa, Canada, May 13-14.

American Broncho-Esophagological Association, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16, (afternoons).

The American Laryngological, Rhinological and Otological Society, Inc., will hold its Annual Meeting at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16-17 (mornings only).

Please make early plans to attend the 1956 Spring Meetings in Canada. Both the Seigniory Club and Montreal present most attractive features for you and your family. More information about the places will be given later.

Reservations at the Sheraton-Mt. Royal Hotel should be made early by addressing the Reservation Supervisor, 1455 Peel Street, Montreal, P. Q., Canada.

POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Michigan, on April 19, 20 and 21, 1956, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology at the University of Michigan Medical School.

Guest lecturers of national prominence, together with members of the staff of the Department of Otolaryngology, will participate in the program.

For further information, write to Dr. John M. Sheldon, Director, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M. D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

PROGRAM 1956

March 12 - 14, 1956

THE DALLAS SOUTHERN CLINICAL SOCIETY

Joint Meeting Fort Worth E.E.N.T. and Dallas O. and O.

GUESTS: Ophthalmology:

Dr. Trygve Gundersen
Dr. Jack S. Guyton

Otolaryngology:

Dr. Franz Altmann
Dr. Paul J. Moses

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seigniory Club, Ottawa, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.
First Vice-President: Robert L. Goodale, 330 Dartmouth St., Boston, Mass.
Second Vice-President: Paul H. Holinger, 700 North Michigan Ave., Chicago 11, Ill.
Secretary: Harry P. Schenck, 326 South 19th St., Philadelphia 3, Pa.
Treasurer: Fred W. Nixon, 1027 Rose Building, Cleveland, Ohio.
Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City, Iowa.
President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Daniel S. Cunning, 115 East 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October, 1956.

THE AMERICAN RHINOLOGIC SOCIETY

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chessen, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois, October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1956.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1956.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Armand L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
General Secretary: Dr. C. E. Muñoz MacCormick, P. O. Box 9111, Santurce 29, Puerto Rico.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: April 8-12, 1956, San Juan, Puerto Rico.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. William J. Hitschler.
Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Paul H. Holinger, 700 No. Michigan, Chicago 11, Ill.
Vice-President: Dr. Jack Allan Weiss, 109 No. Wabash Ave., Chicago 3, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1956.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Frederick A. Holden, Medical Arts Building, Baltimore 1, Maryland.
Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
Vice-Chairman: Dr. William J. G. Davis, 1632 K Street, N. W., Washington 6, D. C.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting: May 26 - June 2, 1956.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradford, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting: George Vanderbilt Hotel, Asheville, N. C., Sept. 16-19, 1956.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Norman Eaddy, Sumter, S. Car.
Vice-President: Dr. J. H. Gressette, Orangeburg, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.
Meeting jointly with the North Carolina Eye, Ear, Nose and Throat Society, George Vanderbilt Hotel, Asheville, N. Car., Sept. 17-18-19, 1956.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. A. R. Hollender, 605 Lincoln Road, Miami Beach, Fla.
Vice-President: Dr. Curtis D. Benton, Jr.
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edwin G. Grafton.
Vice-President: Dr. L. A. Furchgott.
Secretary-Treasurer: Dr. Hal W. Maxwell.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Robert A. Norene, M.D.
Secretary-Treasurer: Leland R. House, M.D.
Chairman of Section on Ophthalmology: Wendell C. Irvine, M.D.
Secretary of Section on Ophthalmology: Stephen J. Popovich, M.D.
Chairman of Section on Otolaryngology: Herschel H. Burston, M.D.
Secretary of Section on Otolaryngology: Ross A. Goodcell, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.
Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Lester T. Jones, M.D., 624 Medical Arts Bldg., Portland (5), Oregon.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: April 15-19, 1956.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Hon. Chairman: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles 57, Calif.
Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. S. Gorrell, 110 Medical Arts Bldg., Calgary, Alta.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: June 6-7, 1956, Chateau Frontenac, Quebec, Canada.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

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Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

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Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

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Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

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SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

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First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
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Vice-Presidente: Dr. D. Jose Perez Mateos.
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Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael Garcia Tapia.

PORTUGUESE OTORHINOLARYNGOLICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida de Liberdade 65, 1° Lisbon.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA

Presidente: Dr. Alberto Luis de Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1.°, Lisboa.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Sufie Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filno Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Apartado Postal 21815
Mexico, D. F.

Honorary President: Dr. Ricardo Tapia y Fernández.
President-Elect: Dr. Miguel Arroyo Güijosa.
Secretary: Dr. Luis Vaquero.
Secretary-Elect: Dr. Benjamin Macias Jiménez.
Treasurer: Dr. Rubén Mingram Camargo.
Meeting: May 5, 1956.

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